Primary central nervous system lymphoma in AIDS: a wider spectrum of CT and MRI findings

Abstract Diagnosis of primary central nervous system lymphoma (PCNSL) in patients with AIDS based on radiological findings is still a challenging problem. Our purpose was to review the CT and MRI findings in PCNSL in our patients with AIDS and compare them with those reported in the literature. CT and MRI of 28 patients with AIDS and pathologically confirmed PCNSL were analysed retrospectively for the number of lesions, their site, size, density, signal intensity, contrast enhancement, oedema and mass effect. We found 82 lesions. On CT 45 lesions were found in 22 patients, whereas MRI revealed 66 in 20 patients. The lymphoma was solitary in 20 patients (29%) and multiple in 20 (71%). Spontaneous haemorrhage was seen in 7 patients. Contrast-enhanced MRI showed no enhancement in 27.3% (18/66) of the lesions. In one patient diffuse signal abnormalities in the white matter were seen on T2-weighted images. Our findings suggest that the previously described spectrum imaging characteristics of PCNSL has widened. Neuroradiologists should be aware of the variable appearance in patients with AIDS. Spontaneous haemorrhage, a non-enhancing lesion, or diffuse white matter changes do not exclude lymphoma in an immunocompromised patient.

Keywords Lymphoma · Brain, tumours · Acquired immunodeficiency syndrome · Computed tomography · Magnetic resonance imaging

Introduction Primary central nervous system lymphoma (PCNSL) is of major importance because of its increasing frequency in both immunocompetent and immunocompromised patients. It has been estimated that up to 6% of all patients with AIDS will develop PCNSL, making AIDS one of the most important risk factors [1]. In AIDS the lymphomas are of high-grade malignancy, and almost always the non-Hodgkin’s type. The disease is progressive in the majority of cases and prognosis remains poor [2].

The appearances of PCNSL on CT and MRI in AIDS have been described in a rather small number of radiological studies [3–8]. Differentiation of PCNSL and toxoplasmosis remains unsatisfactory with both modalities. Because of the decreasing frequency of cerebral toxoplasmosis and the rise in PCNSL, new diagnostic strategies are crucial in patients with AIDS who present with focal brain lesions.

We performed a retrospective analysis of 28 human immunodeficiency virus (HIV) seropositive patients with documented PCNSL, to review our experience with imaging of PCNSL in our AIDS patients, to define the spectrum of findings which could suggest PCNSL at an earlier stage, and to compare our imaging findings with those reported in the literature.
Methods

The records of neuropathologically examined autopsies and biopsies of patients with AIDS were reviewed and patients with pathologically confirmed PCNSL identified. CT and/or MRI were available for 28 patients: 22 had CT (including 22 contrast-enhanced studies) and 20 had MRI (including 18 contrast-enhanced studies).

There were 26 men and two women, aged 22–50 years (mean 38.3 years). There were 14 patients who underwent stereotactic biopsy, in 19 cases autopsy was performed, and in six cases both biopsy and autopsy data were available. Eight patients had CT only, six patients had MRI only, and 14 patients underwent both. CT was performed both before and after intravenous contrast medium, with axial 3–5 mm sections for the posterior cranial fossa and 8–10 mm-thick sections for the cerebrum. Nonionic contrast medium was given, 1 ml/kg body weight.

MRI was performed on 1.5, 1.0 or 0.5 T units. Pulse sequences used were: T1-weighted spin-echo (SE) (20 patients), T2-weighted fast SE (20 patients) and fluid-attenuated inversion-recovery (FLAIR) (8 patients). Gadopentetate dimeglumine was given as an intravenous bolus (0.1 mmol/kg body weight).

The images were reviewed independently by two experienced reviewers (M.M. Thurnher, E. Schindler), with regard to the number of lesions, their site, size, density (CT) or signal intensity (MR), the pattern of contrast enhancement (solid, ring, irregular/sinusoid) oedema and mass effect; oedema and mass effect were graded as mild, moderate or marked.

The medical records were also reviewed for coexistent CNS pathology and lymphoma outside the CNS.

Results

We detected 82 lesions (mean 2.9/patient) in 28 patients. Lymphoma was solitary in eight patients (29%) and multiple in 20 (71.4%). The basal ganglia were the most frequently affected part of the brain, with lesions in this area in 15/28 patients (53.6%). Lesions were seen in the frontal lobe in 12 patients (43%), parietal lobe in eight (29%), temporal lobe in seven (25%) and occipital lobe in four (14%). One patient had lesions in thepons (3.6%). Both infra- and supratentorial lesions were seen in five patients (18%); no patient had lesion limited to the posterior cranial fossa. The corpus callosum was involved in six patients (21.4%), and periventricular lesions were seen in five (18%).

On CT, we saw 45 lesions in 22 patients and on MRI 66 in 20 patients. Of the 45 lesions seen on CT, six lesions were isodense, 36 of low density, and three of hyperdense on unenhanced images. Seven lesions (15.5%) showed homogeneous increased density, five (11%) showed rim and 33 (73%) irregular peripheral enhancement.

On T1-weighted MRI, the lesions were isointense or gave low signal and no lesion showed high signal. On T2-weighted images, six lesions gave low signal, whereas the remaining 60 were of high signal. All showed high signal on FLAIR images (Fig. 1). Contrast-enhanced T1-weighted images were available for 18/20 patients; no lesion showed homogeneous enhancement; nine lesions showed ring and 38 irregular peripheral enhancement.

There were 18 lesions (27%) which showed no contrast enhancement on MRI: three lesions in the right temporal lobe, seven in the frontal cortex or subcortical region, five in the basal ganglia, one in the pons and two in the periventricular white matter (Fig. 2). Diffuse high signal was seen on T2-weighted images in the white matter of one patient; these diffuse changes showed no enhancement (Fig. 1).

The size of the lesions varied considerably from 0.8 cm to 10 cm (mean 1.8 cm). Mass effect was mild in 11/28 patients (39%), moderate in seven (25%), and significant, with midline shift, in seven (25%). Oedema was mild in nine patients (32%), moderate in nine (32%), and marked in seven (25%); in three patients (11%), neither oedema nor mass effect was seen.

Subacute hemorrhage (areas of high signal on T1-weighted images) was detected within the tumours in seven patients (25%) (Fig. 3).

Of there 28 patients with primary cerebral lymphoma, 4 had lymphoma in organs outside the CNS: two had gastric lymphoma; in one lymphomatous infiltration of the kidney and pancreas was diagnosed postmortem, and one patient had lymphoma of the lung.

There were two patients who also had cerebral toxoplasmosis, with toxoplasmosis sequelae found at autopsy. In one patient cerebral lymphoma and toxoplasmosis were present simultaneously. In three patients, cytomegalovirus ventriculitis was diagnosed at autopsy. One patient had cryptococcal meningitis and cortical haemorrhages.

Both CT and MRI were performed in 14 patients at intervals of 0 to 7 days (mean 2 days). MRI demonstrated more lesions than CT in six, 36, compared to 13. Of the 23 lesions not detected with CT, seven were frontal, five parietal, three temporal, one periventricular, four in the basal ganglia, one in the pons and two others in the posterior cranial fossa. In one patient CT showed no abnormality, while MRI revealed multiple lesions. In three patients a solitary lesion was seen on CT, and multiple lesions were demonstrated on MRI. In eight patients CT and MRI provided the same information; CT was never superior to MRI. CT was not performed in the patient in whom MRI showed diffuse white-matter abnormalities.

Discussion

After cerebral toxoplasmosis, primary central nervous system lymphoma is the second most common cause of cerebral mass lesions in AIDS, and occurs in about 3–18% of cases [1]. These high-grade malignant tu-